THE PLANT DISEASE REPORTER

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THE PLANT DISEASE SURVEY

DIVISION OF MYCOLOGY AND DISEASE SURVEY

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CEPHALOSPORIUM WILT OF PERSIMMON IN THE SOUTHEAST

Bowen S. Crandall

The American persimmon, Diospyros virginiana, is a tree of considerable economic importance. Over most of its range (Fig. 1) its fruits are used as food by man and by domestic animals, and are a very valuable source of food for fur-bearing animals and other wild game. The tree itself is valued by the soil conservationist, since by its growth in thickets it tends to anchor the soil and prevent washing and thus preserves the top soil which may otherwise be lost. When cultivated land is abandoned for crops the persimmon is often among the first of the wild trees to appear. Persimmon lumber also has a high value. In utilization it is classed with dogwood, the supply of which has already been greatly reduced. Annually two million feet of persimmon are used for the manufacture of golf stick heads and shuttles.

In 1937 a hitherto unrecognized disease of the native persimmon was reported from Tennessee[1,2]. While the disease behaves as if it were introduced, it has not yet been possible to relate it to any similar trouble of persimmon described as occurring in foreign countries. The Japanese persimmon, D. kaki, seems resistant but may be affected when grafted on roots of the native persimmon. One instance is known in Florida where the disease apparently spread into a Kaki orchard on American roots from adjacent infected native persimmons.

The disease is a rapidly killing wilt. Two months is sometimes enough for the infection and death of the whole tree. The external symptoms are those typical of a vascular infection. On trees already severely infected only small chlorotic leaves appear in the spring. Such trees usually die by midsummer. Infected trees that bear normal leaves in the spring or trees that become infected during early spring develop flags of yellow foliage in their tops, soon followed by general wilting and discoloration, upon the advent of hot weather.

An unidentified species of Cephalosporium has been isolated from the dying trees, and the disease has been produced in healthy persimmon trees inoculated with this fungus[3].

1/ Crandall, Bowen S. and Ross W. Davidson. Cephalosporium wilt of persimmon. Plant Disease Reporter 21: 251-252. July 15, 1937.

2/ Crandall, Bowen S. Persimmon wilt. Plant Disease Reporter 21: 338. Oct. 1, 1937.

3/ Beattie, R. Kent and Bowen S. Crandall. Disease attacks the per- persimmon. Amer. For. 45: 120-121, 124. 1939.

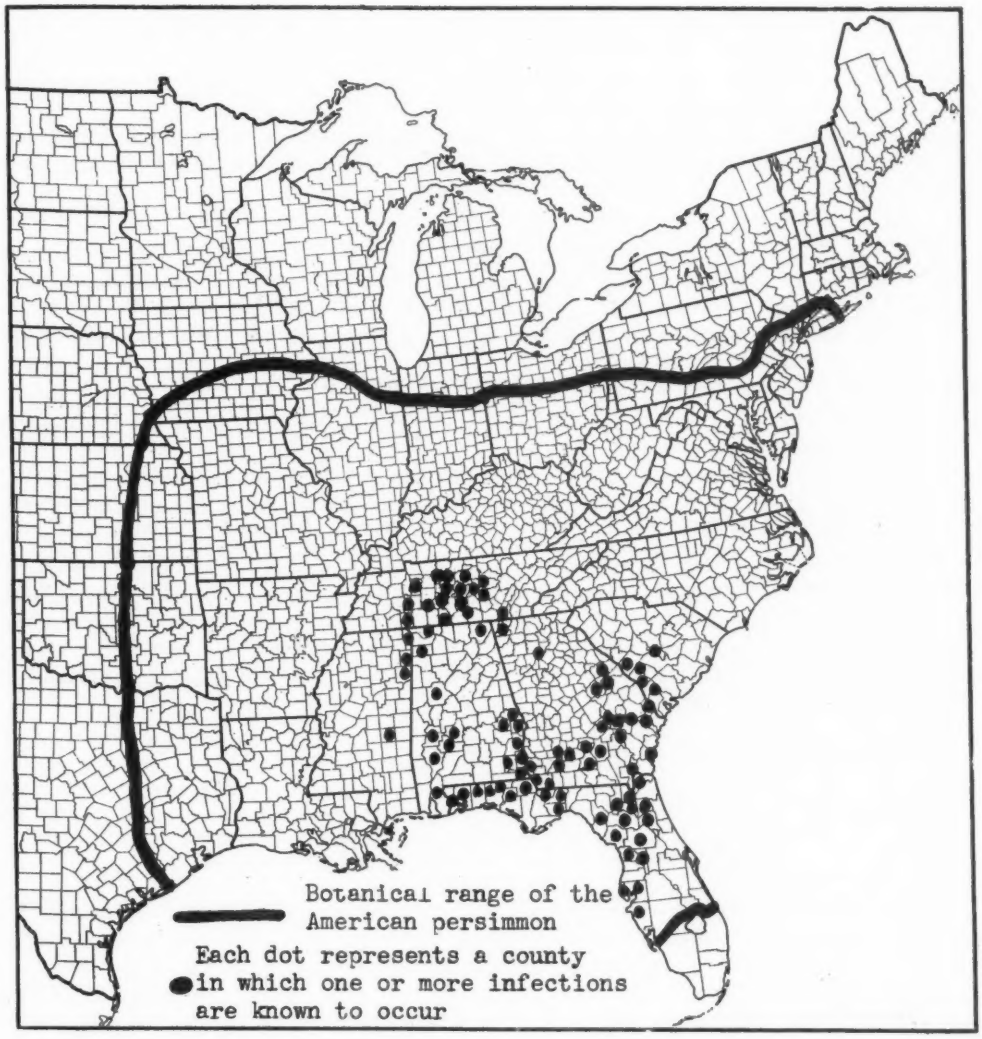

Botanical range of the
American persimmon
Each dot represents a county
in which one or more infections
are known to occur

Figure 1. Areas known to be infected by persimmon wilt at the close of the 1938 scouting season.

An infected tree is characterized by fine brownish-black streaks scattered throughout the more recent annual rings. After a tree dies the fungus fruits under the bark, which soon loosens, and also in cracks on the bark of the dying or dead tree. Under the loose bark the masses of salmon-colored spores are often so abundant that they can be scooped up with a teaspoon. These spores appear to be wind-disseminated in large quantities.

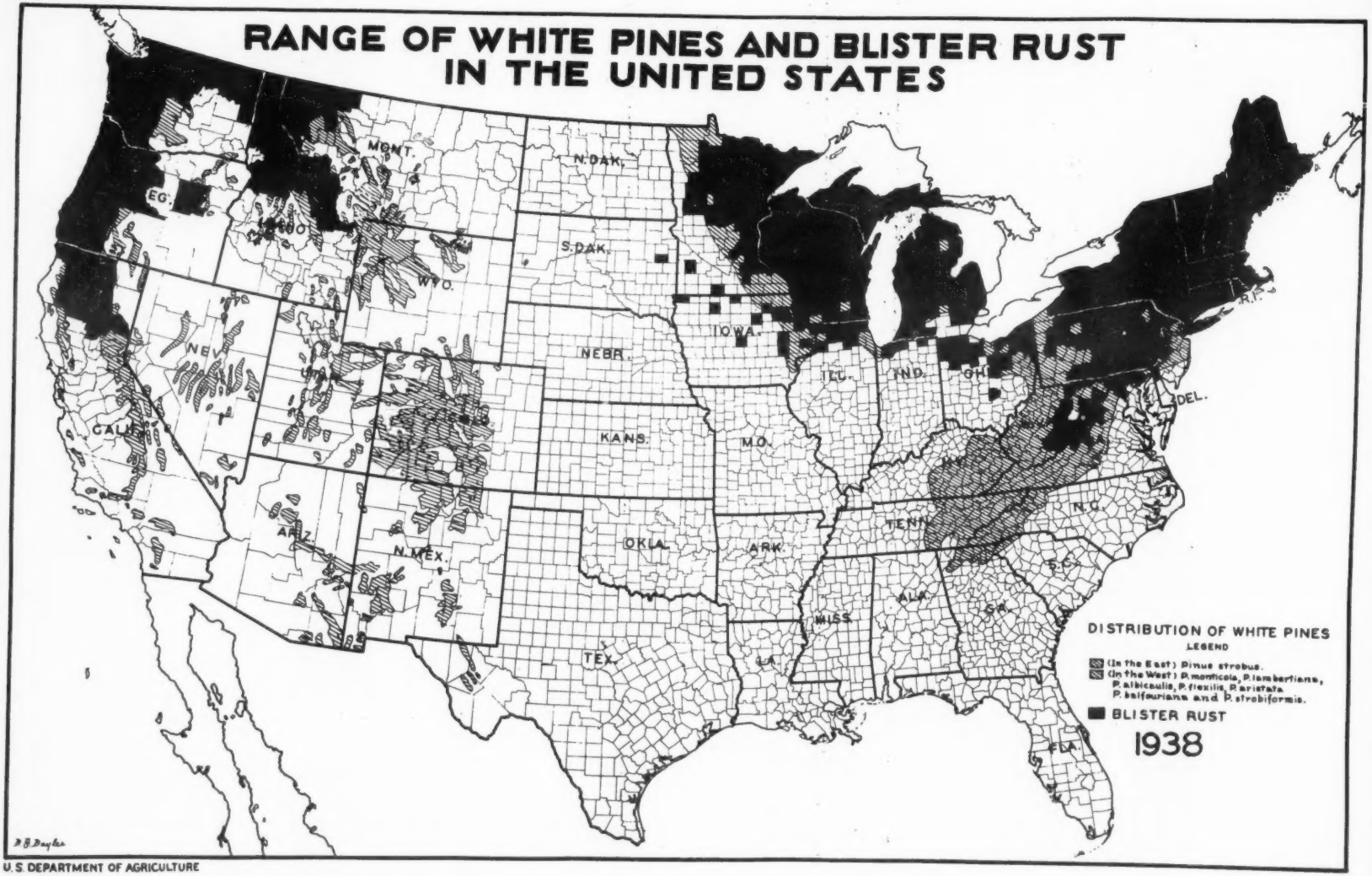
This dying of the American persimmon was first noted by a resident of Tennessee in 1933 or 1934 in a single tree, but when reported to the Division of Forest Pathology in 1937 it had also appeared in the other scattered, nearby persimmon trees. On this farm, where the disease has been under observation for two seasons, only isolated individuals comprising about 5 percent of the original stand now remain alive. Search late in 1937 showed infections present in six middle Tennessee counties. A survey of the Southeastern States during the summer of 1938 revealed infections scattered through the central basin of Tennessee, eastern Mississippi, and portions of Alabama, Georgia, Florida, and South Carolina (Fig. 1). In Tennessee, west Florida, and north central Florida were found centers of infection dating at least as far back as 1936. Here and elsewhere it is impossible to obtain evidence, in the form of dead trees, to show whether the disease was present earlier than 1936 because wilt-killed trees are attacked and often completely rotted in less than two years by Schizophyllum commune. Sporophores of this fungus are commonly found on wilt-killed trees a few months after their death.

(Division of Forest Pathology, Lebanon, Tennessee).

SPREAD OF WHITE PINE BLISTER RUST IN 1938

The Division of Plant Disease Control of the Bureau of Entomology and Plant Quarantine each year during the latter part of the summer makes a survey to determine the extent to which white pine blister rust [Cronartium ribicola] has apparently spread to new localities. In connection with these surveys many more Ribes than pines are examined, since infection is much easier to find on the currant and gooseberry plants, and Ribes infection is often discovered many miles in advance of the farthest known spread of the disease on pines. Later, of course, infection may be expected on pines in the same localities as a result of the spread of sporidia from the Ribes.

The information thus obtained is important in connection with planning the priority of white pine blister rust control operations. Ribes eradication is carried out around local pine stands of sufficient value to justify the expense of protection, and such protection is effective regardless of the extent of blister rust infection in the vicinity. Blister rust control


RANGE OF WHITE PINES AND BLISTER RUST
IN THE UNITED STATES
MONT.
N.DAK.
S.DAK.
WYO.
NEBR.
IOWA
ILL.
IND.
OHIO
KANS.
MO.
OKLA.
ARK.
TEX.
MISS.
ALA.
GA.
FLA.
S.C.
N.C.
TENN.
ARIZ.
N.MEX.
DEL.
R.I.
DISTRIBUTION OF WHITE PINES
LEGEND
(In the East) Pinus strobus.
(In the West) P. monticola, P. lambertiana, P. albicaulis, P. flexilis, P. aristata, P. balfouriana and P. strobiformis.
BLISTER RUST
1938
U.S. DEPARTMENT OF AGRICULTURE

work accordingly is not primarily directed at retarding the spread of infection to new localities through wind carriage of the aeciospores, but is carried out to prevent injury to valuable pine stands by blister rust after the disease arrives in a locality.

During the calendar year 1938, blister rust was found for the first time on either white pine or Ribes in 70 counties. Six of these counties are in the Southern Appalachian States, 60 in the North Central States, 2 in Montana, and 2 in California. This information is shown below in detail:

Counties in Which Blister Rust was Found for the First Time on White Pines or Ribes in 1938 by States and Regions.

Region	State	County	Infection Found on Pine	Infection Found on Ribes	Host Plant	Month Found
Southern Appalachian	Virginia	Greene		X	R. rotundifolium	September
"	"	Warren		X	"	October
"	"	Rockbridge		X	"	"
"	"	Shenandoah		X	"	"
"	"	Highland	X		P. strobus	February
"	West Virginia	Hardy		X	R. cynosbati	September
North Central	Ohio	Knox	X		P. strobus	April
"	"	Ashland	X		"	"
"	"	Ashland		X	R. nigrum	October
"	"	Wyandot		X	"	November
"	"	Sandusky		X	"	"
"	"	Seneca		X	"	"
"	"	Holmes	X		P. strobus	May
"	"	Holmes		X	-	August
"	"	Allen		X	R. nigrum	October
"	"	Defiance		X	"	"
"	"	Henry		X	"	"
"	"	Lucas		X	"	"
"	"	Paulding		X	"	"
"	"	Putnam		X	"	"
"	"	Williams		X	"	"
"	"	Wood		X	"	"
"	"	Hancock		X	"	November
"	Indiana	Porter		X	"	October
"	"	St. Joseph		X	"	"
"	Illinois	Stephenson		X	"	September
"	"	Whiteside		X	"	October

Region	State	County	Infection Found on Pine	Infection Found on Ribes	Host Plant	Month Found
North Central	Illinois	Ogle		X	R. nigrum	October
"	"	DeKalb		X	"	"
"	Iowa	Butler		X	"	"
"	"	Clayton		X	"	"
"	"	Clinton		X	"	September
"	"	Winnebago		X	"	"
"	"	Chickasaw		X	"	November
"	Michigan	Chippewa	X		P. strobus	July
"	"	Montmorency	X		"	April
"	"	Branch		X	R. vulgare	November
"	"	Wexford		X	R. cynosbati	August
"	"	Kalamazoo		X	"	November
"	"	Lake		X	"	August
"	"	Berrien		X	R. nigrum	October
"	"	St. Joseph		X	R. vulgare	"
"	"	Eaton		X	R. americanum	"
"	"	Barry		X	R. cynosbati	"
"	"	Crawford		X	R. hirtellum	"
"	"	Kalkaska		X	R. cynosbati	September
"	"	Ionia		X	"	August
"	"	Van Buren		X	R. nigrum	November
"	"	Grand Traverse	X		P. strobus	September
"	Wisconsin	Columbia	X		"	April
"	"	Juneau	X		"	June
"	"	Juneau		X	R. cynosbati	"
"	"	Calumet		X	"	August
"	"	Green Lake		X	"	"
"	"	Iowa		X	R. nigrum	September
"	"	Rock		X	"	"
"	"	Crawford		X	"	October
"	"	Dodge		X	R. nigrum & sativum	"
"	"	Green		X	R. nigrum	"
"	"	Marquette		X	R. americanum	"
"	"	Ozaukee		X	R. nigrum	"
"	"	Richland		X	"	"
"	"	Washington		X	"	"
"	Minnesota	Mahnomen		X	R. aureum	"
"	"	Ottertail		X	R. cynosbati	"
"	"	Hennepin		X	R. nigrum	"
"	"	Ramsey		X	"	"

Region	State	County	Infection Found on Pine	Infection Found on Ribes	Host Plant	Month Found
North Central	Minnesota	Dakota		X	R. nigrum	October
	"	Goodhue		X	"	"
Western White Pine	Montana	Flathead		X	R. inerme and R. viscosissimum	September
"	"	Glacier		X	R. inerme and R. viscosissimum	October
Sugar Pine	California	Butte		X	R. roezli	September
"	"	Plumas		X	"	August

In the Southern Appalachian States the rust was found on white pine in Highland County, Virginia and on Ribes in 4 counties in Virginia, and one in West Virginia. These discoveries did not extend the spread of the disease further south, but added new counties within the previously known limits of infection in the Southern Appalachian Region.

The most extensive spread of the disease into new territory occurred in the North Central States where weather conditions probably were more favorable for the dissemination of the rust. Infection was found for the first time on white pine in 8 counties in Ohio, Michigan, and Wisconsin, and on Ribes in 55 counties in the 7 States comprising the North Central blister rust control region. In 38 of the 55 counties infection occurred on the cultivated black currant, Ribes nigrum. The large number of counties in which the disease was found on cultivated black currants shows the importance of this species as a disseminator of the rust and the need for the eradication of Ribes nigrum in regions where the white pines are valuable forest and ornamental trees. The southern limits of the known infected area have been extended during the past year from one to three counties in Ohio, Indiana, Illinois, and Iowa. In addition, many newly infected counties were added within the previously known limits of spread.

In Montana the disease was found to be widely distributed on wild Ribes on both sides of the Continental Divide. Infection was found at 5 different points where inspections were made in Glacier National Park. This represents an eastward extension of the rust into Flathead and Glacier Counties. Glacier National Park is situated within these counties.

Rust infection on Ribes in California shows that the disease spread southward another 35 miles during 1938, and is now present about 160 miles below the Oregon-California border. Numerous other infections on Ribes

were found within the sugar pine area previously reported as infected. No rust was found on sugar pine, as it takes from 3 to 4 years after infection for the resulting cankers to become easily discernible on pines, and sufficient time has not elapsed since discovery of the disease in the State to enable the field men to find it readily on sugar pine. However, it is very probable that scattered pine infections exist in northern California.

One of the most significant finds occurred in Shasta County about 80 miles south of the Oregon line. In 1937 scouting activities in this locality uncovered but two infected Ribes, each with but one leaf infected. A year later 300 infected Ribes, 50 of them Ribes nevadense, and the remainder R. roezli, were found to be heavily infected in the same vicinity.

Again in Tehama County west of Mt. Lassen, 1937 scouting showed no infection while in 1938 over 100 infected R. roezli, R. inerme, and R. nevadense bushes were found in a small localized area. Such discoveries, both in good association with sugar pine, have laid the foundation for local infection on pines within the next few years.

(Division of Plant Disease Control, Bureau of Entomology and Plant Quarantine).

FUSICLADIUM ROBINIAE AND MACROSPORIUM SP. IN FOREST TREE NURSERIES

William C. Davis and Ross W. Davidson

Fusicladium robiniae Shear is associated with leaf spots and petiole lesions on black locust seedlings in some forest tree nurseries. The discolored spots, the diameters of which vary from about 0.5 to approximately 2.0 millimeters, have dark definite margins and light-colored centers. The petiole lesions, which may be 5 or more millimeters in length, are one-sided to girdling; their long axis is parallel to that of the petiole. The lesions are generally somewhat shrunken, and somewhat darker in color at the margin than at the center.

In several instances death of black locust seedlings has been attributed to Fusicladium robiniae. Frequently premature defoliation, and in a few instances stunting of seedlings, are associated with infection by this organism. The fungus may attack young seedlings, after killing or stunting them, but it usually does not make its appearance until the stock is somewhat older, the greatest intensity of the disease condition centering in August to September.

The macrosporium leaf disease was first reported on locust in the

United States by Miller[1/] in a tree seedling nursery at Athens, Georgia. Untreated seed from Austria was used at this nursery, and it has been suggested that the fungus might have been introduced. Correspondence with Miller shows that the disease recurred at Athens in 1936, but was not observed in 1937. The spots on the leaflets associated with this fungus are light brown and characterized by concentric circles and small dark sporulating masses of the fungus. These spots are larger (about 3-8 millimeters) than those associated with Fusicladium robiniae. This macrosporium leaf disease does not appear to be causing any actual loss of seedlings in nurseries.

Control measures have not been developed for either of these diseases, but sprayings with Bordeaux may be expected to give some measure of control.

The observations presented in the tabulation indicate that Fusicladium robiniae is widely distributed, occurring in nine states, while Macrosporium sp. is more restricted in its distribution.

Summary of observations relative to Fusicladium robiniae and Macrosporium sp.

State and nearest town	Date observed	Approximate age of seedlings	Proportion of stock affected
Alabama:			
Muscle Shoals a/	Sept. 22, 1938	4 months	very small
Thorsby a/	Sept. 21, 1938	3 months	small
Arkansas:			
Lurton	Oct. 19, 1938	3-5 years	moderate b/
Georgia:			
Americus a/	Sept. 20, 1938	4 months	moderate
Flowery Branch a/	Sept. 19, 1938	4 months	moderate
Maryland:			
Beltsville	Sept. 3, 1937	3 months	none
	Aug. 19, 1938	2 1/2 months	none
Missouri:			
Licking a/	Oct. 18, 1938	15 months	large b/

1/ Miller, J. H. Forest seed bed diseases in Georgia and South Carolina. Plant Disease Reporter 19: 259, Oct. 1, 1935. (Since the fungus causing the disease was originally referred to Macrosporium, this name is used pending further study).

State and nearest town	Date observed	Approximate age of seedlings	Proportion of stock affected
North Carolina:			
Bryson City a/	Sept. 29, 1938	4 months	moderate
Chapel Hill	Sept. 9, 1938	3 months	moderate
Clayton	June 13, 1938	1 week	none
	Sept. 10, 1938	4 months	small
Hendersonville	Aug. 15, 1938	1 1/2 months	moderate
	Sept. 30, 1938	3 months	large
South Carolina:			
Camden	Sept. 15, 1938	5 months	moderate
Whitmire	June 16, 1938	1 year	large
	Sept. 17, 1938	1 1/2 years	moderate
Rock Hill	Sept. 16, 1938	4 months	moderate
Tennessee:			
Clinton a/	Sept. 27, 1938	4 months	moderate
Jackson	Oct. 24, 1938	4 months	moderate b/
Lebanon	Sept. 24, 1938	15 months	small
Norris Dam a/	June 26, 1938	2 years	small
	Sept. 27, 1938	3 months	none
Pikeville	June 24, 1938	3 weeks	moderate
	Sept. 26, 1938	4 months	large
Virginia:			
Charlottesville	Oct. 1, 1937	3 months	moderate
	Aug. 2, 1938	1 month	small
	Sept. 7, 1938	(2 1/2 months	small
		(2 years	large
Gretna	Oct. 1, 1937	4 months	large
	Aug. 3, 1938	2 1/2 months	none
	Sept. 7, 1938	3 1/2 months	moderate

a/ Both fungi observed; all other localities for F. robiniae only. Relative amount of Macrosporium-affected stock small at all locations.

b/ Collected by D. H. Latham.

(Civilian Conservation Crops and Division of Forest Pathology, Bureau of Plant Industry, cooperating).

DISEASES OF FRUITS AND VEGETABLES ON THE NEW YORK MARKET DURING THE MONTHS OF OCTOBER, NOVEMBER, AND DECEMBER, 1938

James S. Wiant and C. O. Bratley

APPLES:

Washing injury on Washington state apples has appeared as frequently during the present season as it did during the same period of the previous year. This has been true despite the fact that the lowering of the spray residue tolerance should have made less severe washing treatments possible. The injury has been largely confined to the calyx cavity, indicating that the fruit was rinsed insufficiently.

Specimens of apples showing severe sulfur dioxide injury were received in late October from a fruit and vegetable inspector at Hartford, Connecticut. The entire surface of the fruits was speckled with small bleached areas ranging from pin-point size to one-eighth inch diameter. It was reported that fumigation with a sulfur preparation had been made for insect control while the apples were in the storage room.

CELERY:

Field freezing injury of considerable importance was noted in a number of carlots of California celery received between late November and mid-December. From few to many of the outer petioles had a glassy watersoaked appearance with the tissues pithy and easily broken apart, and the epidermis in part wrinkled and broken.

An unusual occurrence of edema was found in early December on samples of California celery sent in by a fruit and vegetable inspector from East Providence, Rhode Island, who reported that 20 to 30 percent of the stalks were affected in some crates of the carlot. The tiny pimple-like swellings occurred in abundance on both the inner and outer surfaces of from few to many of the outer petioles per stalk, erupting through the epidermis and turning dark as they developed.

GRAPES:

Sulfur dioxide injury was about as prevalent on California grapes this season as it has been in recent years. In only two or three shipments, however, was the injury noted in all lugs in the car.

OLIVES:

Sulfur dioxide injury characterized by extensive surface pitting and bleaching was noted in a number of lots of green olives that were received from California in mixed cars of olives and grapes. Gas used for control of mold on the grapes was responsible. One receiver had five cars so affected during a single week.

Several lots of California green olives (totaling 350 lugs) showed from 25 to 80 percent internal breakdown when removed from storage where they had been held at 34° F. for three weeks. The injury ranged from a moderate to a severe and complete browning of the flesh accompanied in the latter case by a brown discoloration of the skin over a portion of the olive.

ONIONS:

A serious amount of small sclerotial neck rot (Botrytis squamosa) was noted in a carlot of white onions received from Ohio in mid-October. Six percent of the onions were affected with a decay at the neck; an additional ten percent had only the outer dry scales affected. The characteristic sclerotia were present in abundance.

In a carlot of Michigan white onions received in late October, from 10 to 95 percent were affected with smudge (Colletotrichum circinans).

ORANGES:

Skin breakdown on de-greened and "color added" oranges was found in small percentages in many shipments of Florida oranges but the trouble was not as prevalent as it has been during the past three or four seasons.

PEAS:

From 30 to 60 percent of the pods in a carlot of California peas received in late November were affected with thrips injury. Field freezing injury of California peas was of importance in late November and early December.

PEARS:

Cladosporium rot (Cladosporium sp.) originating at stem punctures was found to the extent of 1, 2, 2, and 5 percent respectively in four carlots of Bosc pears arriving in early December from Placerville, California. This is the same shipping point from which several carlots were received last season in the same condition.

PEPPERS:

Two carlots of California peppers arriving at New York about the first of November contained 14 percent and 30 to 100 percent decay most of which consisted of scattered spots of gray mold rot (Botrytis sp.).

POMEGRANATES:

As high as 10 percent of the fruit was found decayed in several carlots of California pomegranates packed in tight cardboard cartons and wooden boxes. Black mold rot (Aspergillus niger), blue mold rot (Penicillium sp.), and an unidentified fungus which produces reddish pycnidia were found to be responsible for the decay.

SQUASH:

Two lots of Texas squash (pattypan type) received by boat (each by a different steamship line) in late November contained 18 percent and 50 percent decay respectively. Apparently little if any regrigeration was given enroute. The decays were chiefly rhizopus rots (Rhizopus sp.) and fusarium rots (Fusarium sp.) - in advanced stages.

TANGERINES:

More than usual amounts of stem-end decay (Phomopsis citri and Diplodia natalensis) were found in Florida tangerines upon arrival at New York City this season.

Usual amounts of skin breakdown caused by injuries from packing house operations were noted on Florida tangerines.

TOMATOES:

California tomatoes arriving during October and November were probably in as poor a condition as they have been during recent years. Uneven coloring, both associated and not associated with mottling from virus diseases was common in the ripening rooms. At the same time the rate at which the tomatoes ripened was slow.

Decays were abundant both at the time of arrival and in the ripening room. Green mold rot (Cladosporium sp.), alternaria rot (Alternaria sp.), macrosporium rot (Macrosporium sp.), and pleospora rot (Pleospora lycopersici) predominated. Of special interest was the unusual prevalence of gray mold rot (Botrytis sp.) in many cases in advanced stages, with half of the individual fruit affected. Late blight rot (Phytophthora infestans) was found in several instances.

Shoulder bruises and other blemishes consisting of skin checking and cracking were likewise abundant.

(Division of Fruit and Vegetable Crops and Diseases).

SYMPTOMS OF BACTERIAL WILT AND ROT (BACTERIAL RING ROT) OF POTATO

In a recent article, the Canadian workers H. N. Racicot, D. B. O. Savile, and I. L. Conners emphasize the potential destructiveness of this "comparatively new, but serious disease" (Bacterial wilt and rot of potatoes--Some suggestions for its detection, verification, and control. Amer. Potato Jour. 15: 312-318. Nov. 1938). They say "The disease appears to be spreading, but its geographical distribution is not completely known; so far it has been reported in widely scattered localities in North America. The discovery of this disease here and there in areas previously thought to be free from it, makes it imperative that every one, pathologist, inspector, and grower, should keep a sharp lookout for it. It has already caused serious losses to growers, and threatens further losses unless it is successfully controlled."

To aid in its detection, they give the following description of symptoms:

"The symptoms appear generally late in the growing season, are very variable, and frequently resemble those of other diseases. The most characteristic ones are here described in the order in which they usually occur. The first symptom is a rolling of the leaflets on affected stems, and is similar to that seen on healthy plants during hot, dry weather. The affected leaflets soon wilt, lose their normal color, and become a dull green. They then feel thin and soft to the touch. The affected leaflets partly or wholly turn yellow, and the chlorotic areas soon die. This necrosis or leaf scorching is chiefly at the margins of the leaflets, and resembles late blight, but is confined to the leaves of individual stems, whereas late blight affects all plants about equally. The ends of the leaf stalks of affected leaves wilt, while the basal parts remain rigid. This resembles tarnished plant bug injury, but in the case of wilt there is no visible injury to the midrib. Eventually the entire leaf dies. One or more stems in a hill may wilt, while the others remain apparently healthy. The stems that wilt are more or less stunted.

"When a plant, with visible symptoms of wilt, is dug, some of the tubers are usually badly diseased, there being, as a rule, every gradation from sound to completely decayed tubers. The bacteria enter the tubers through the stolon and spread along the vascular ring, causing in that region a rot which is creamy, yellow, or light brown in color, crumbly or

cheesy, and odorless. It does not resemble what is generally called 'net necrosis', although it is possible for both bacterial wilt and rot and net necrosis to occur in the same tuber. When the bacteria have spread through the tuber to an eye or eyes, the tissues in that region are often killed, rendering the tuber liable to invasion by soft-rotting organisms. Indeed, many of the tubers are invaded in this manner. The epidermis of badly rotted tubers may become cracked or show reddish discolored areas. In diseased tubers that have escaped invasion by secondary organisms, the rot may become sufficiently advanced to show as a band of decay involving the vascular ring and adjacent tissues."

They discuss overwintering and dissemination, remarking that "There is apparently very little spread of the disease in the field", and control, of which the only recommended means so far is the exclusive use of disease-free seed, and emphasize the importance of prompt and correct diagnosis, as follows:

"Since the symptoms of bacterial wilt and rot appear late in the growing season, are variable and often confused with those of other diseases, and the presence of late blight and insect injury make it increasingly difficult to detect diseased plants, this disease may occur for some time and even become widespread in a locality before it is definitely recognized. Therefore, it is important that bacterial wilt be promptly and correctly diagnosed. A sharp lookout should be kept by every one for this disease. Any case found in a new locality should be verified by a microscopic examination of smears from diseased plants. In doubtful cases, specimens of vines and tubers should be sent for examination to the nearest plant pathological laboratory."

They give directions for making smears from vines and tubers, and for collecting and mailing specimens, with the final warning "Attention may be drawn to the fact that in making inspections and collecting specimens, care should be exercised to avoid the contamination of shoes and clothing by coming in contact with decayed tubers, and in this manner prevent the carrying of the bacteria to other fields. The hands and knives used in handling and cutting diseased tubers should be thoroughly washed between inspections."

These authors are apparently not yet convinced that the disease in North America is really identical with the bacterial ring rot previously known in Europe, caused by Bacterium sepedonicum, as reported by Walter H. Burkholder. Burkholder's article and other information concerning the cause, occurrence, and known distribution of the disease on this continent have been summarized in the Reporter (PDR 22: 444-445).

SMUTTY WHEAT RECEIPTS CONTINUE DECLINE

Bert W. Whitlock

We really thought we had "touched bottom" when we reported last year[1/] that the percentage of smutty-wheat receipts at Columbia River, Puget Sound, and Spokane markets had dropped to about 8 percent from a high point of 36.6 percent in 1931. The 1938 figures indicate, however, that another new low mark will be established.

The August carlot inspections in Oregon and Washington show that the percentage of the 1938 wheat crop that grades smutty will be down to a point somewhere between 5 and 6 percent.

The accompanying chart prepared by E. N. Bates from figures compiled by R. L. Baldwin shows the smut trend since 1930 (Fig. 1).

The downward trend of this curve has continued long enough, we believe, to discount any argument that winter killing has been responsible for the reduction in the smut. Our opinion is that the development or introduction of smut-resistant varieties has had most to do with the improvement, and that seed treatment is a second and equally important factor for the gains that have been made.

We still grow varieties that are susceptible to smut, and we hope that these figures will not cause anyone to believe that the treatment of seed for smut may be relaxed.

We are more or less familiar with the losses due to smut when the wheat arrives at the terminal markets. The discounts applied multiplied by the amount of smutty wheat would give a graphic picture of the size of these losses. If these figures could be added to those that might be prepared on the losses in the field due to smut, the full effect of the work

Years	Cars Smutty (Percent)		Years	Cars Smutty (Percent)	
	For year	For August		For year	For August
1930	28.6	26.9	1935	23.4	23.4
1931	36.6	40.5	1936	16.7	19.2
1932	23.0	27.0	1937	8.2	7.9
1933	22.0	24.2	Mean.	22.3	23.9
1934	20.0	22.2	1938	Est. 5.5	5.9

1/ Plant Disease Reporter 21: 407-409. Dec. 1, 1937.

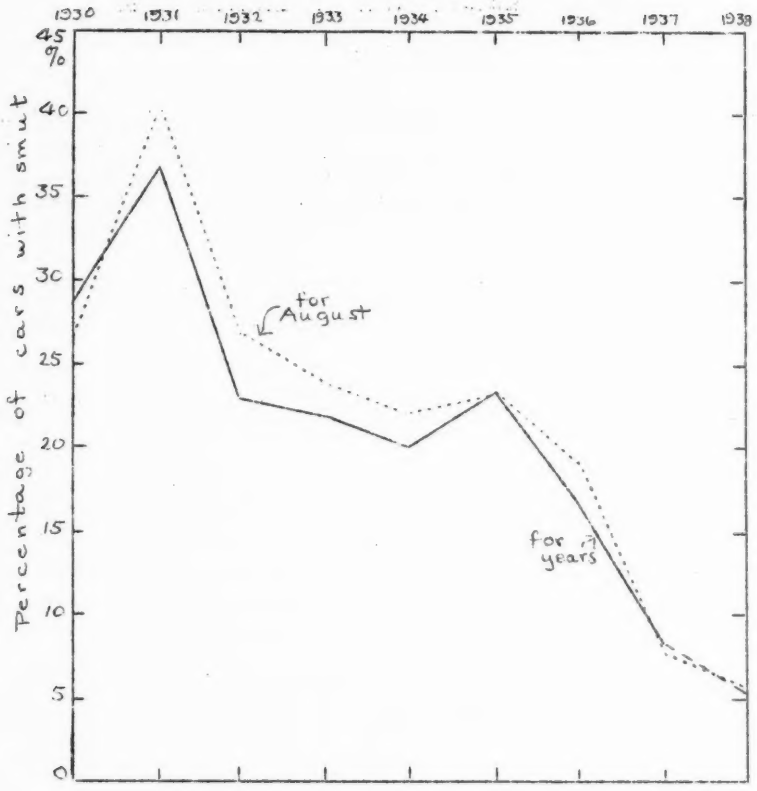


Figure 1.--Pacific Northwest: Percentage of car receipts of wheat grading smutty at Spokane, Washington, and at Columbia River and Puget Sound inspection points, 1930-37, and for month of August for years 1930-38; and on the basis of the mean values an estimate for the year 1938 of 5.5 percent.

of the plant breeders and disease-control officials could be appraised. In our opinion it has been an outstanding piece of crop-improvement work.

(Pacific Coast Headquarters, Federal Grain Supervision, Portland, Oregon. Dec. 28. From Extension Pathologist, no. 35, pages 4-6. Jan. 1939).

SMUTTY WHEAT TRENDS DOWNWARD ON THE MINNEAPOLIS MARKET

R. J. Haskell

The percentage of carloads of smutty wheat received at Minneapolis has been steadily declining since 1930, when a high point was reached with 17.8 percent of all inspected receipts grading smutty. During 1937 only 1.6 percent were smutty, and in September 1938, which is a fairly good index for the year, the percentage was still further reduced to 1.1 percent.

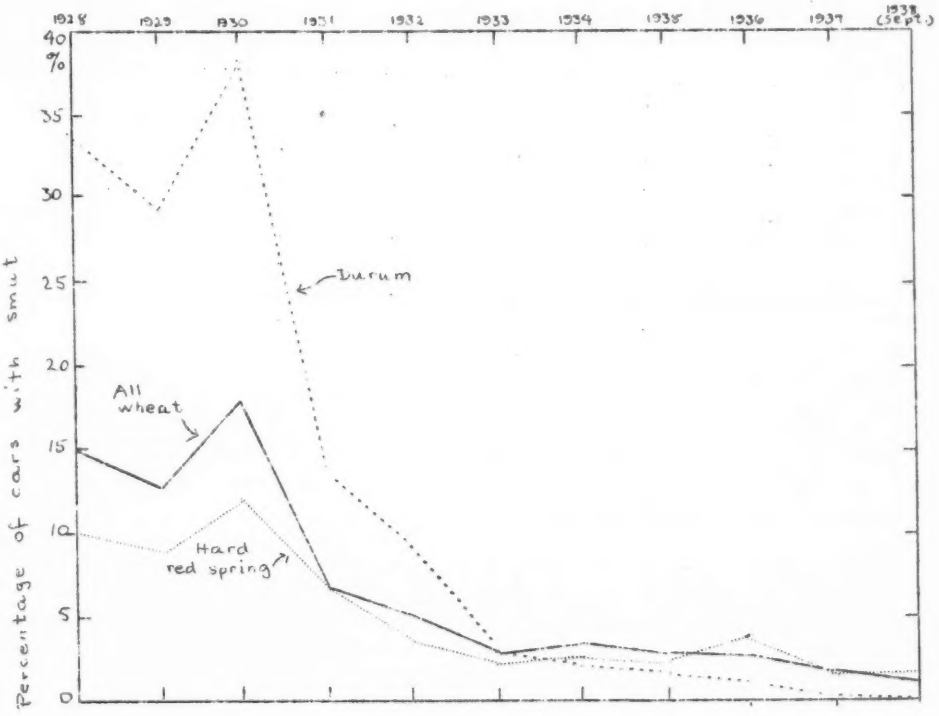


Figure 1.--Percentage of inspected cars of wheat, grading smutty, received at Minneapolis, 1928-37 and for September 1938.

Most of the wheat received at Minneapolis is of the hard red spring class with some durum and hard red winter. Only a very few cars of soft red winter and white wheats are received, these coming from outside the spring-wheat area. The hard red spring, durum, and mixed classes therefore

for the most part represent wheat grown in the States of Minnesota, North Dakota, South Dakota, and Montana.

Percentage inspected cars of wheat received at Minneapolis grading smutty, 1928-37 and September 1938.

Year	Percentage of Cars with Smut						
	Hard Red Spring	Durum	Hard Red Winter	Soft Red Winter	White	Mixed	Total all classes
1928	9.7	33.5	14.1	0	0.9	24.9	14.9
1929	8.7	29.1	7.8	12.5	1.3	25.4	12.6
1930	12.0	37.9	10.9	50.0	1.4	38.1	17.8
1931	6.5	13.6	1.8	100.0	0	19.6	6.7
1932	3.6	9.3	5.8	0	0.8	18.1	5.1
1933	2.1	3.0	4.7	54.8	2.7	9.4	2.9
1934	2.4	2.2	5.7	9.4	3.3	7.2	3.4
1935	2.2	1.7	6.2	33.7	6.1	3.1	2.9
1936	3.7	1.2	1.7	7.7	2.1	3.8	2.8
1937	1.5	0.2	1.1	5.5	0.2	2.2	1.6
Sept. 1938	1.3	0.2	11.0	100.0	- -	2.2	1.1

The decline in the amount of smut in durum wheat is remarkable, dropping from a high of 37.9 percent in 1930 to a negligible low of 0.2 percent in 1938. Less spectacular, but nonetheless positive, are the drops from 12 percent of smut in hard red spring wheat, in 1930, to 1.3 percent in 1938; and from 38.1 percent of smut in mixed wheat, in 1930, to 2.2 percent in 1938 (Fig. 1).

Undoubtedly several factors have contributed to this reduction in smut. A study of weather relations, especially during the critical 1930-1931 period and during the drought years would undoubtedly throw considerable light on the situation, also intensive extension work on seed treatment with better materials and methods is not to be overlooked.

(Extension Plant Pathologist, U. S. Department of Agriculture. Jan. 10. From Extension Pathologist No. 35, pages 6-7. Jan. 1939).

SIX-YEAR STUDY SHOWS WHEN TO EXPECT FREEZE LOSSES IN WINTER WHEAT

(From U. S. Dept. Agr. Press Release, Feb. 19).

Six years' study of winter hardiness in wheat on the Nebraska plains helps to explain why winter wheat is sometimes killed by cold weather and sometimes not. The results do not indicate any practical remedy for winter killing but they do show when crop losses from freezing are to be

expected. The work was cooperative between the Nebraska Agricultural Experiment Station and the Bureau of Plant Industry, United States Department of Agriculture.

Most wheat farmers know that the condition of wheat when cold weather comes on determines to a considerable degree its ability to survive. But, beyond this, little information has been available.

The men who made the study found that winter wheat goes through three definite stages each fall and winter. The first is one of accumulating food reserves, usually ending in late December. These reserves are believed to play a vital role in protecting the plants from subsequent freezing temperatures. The accumulation of these reserves is aided during this period by sunlight and temperatures favorable for growth and is interfered with by frequent rains and cloudy weather.

The second period is one of near dormancy and increased winter hardiness induced by low temperatures. The third period is one of declining hardiness and progress toward active growth which usually takes place no earlier than late January, and often later, depending on temperature.

The periods must occur in the sequence indicated to insure hardiness. Thus, if conditions are not right in the first period for the accumulation of reserves, the wheat may not be able to acquire a sufficient degree of hardiness during the second period to survive very low temperatures. If the weather is too warm during the second period the wheat will not become hardened and may be severely damaged by subsequent cold weather.

BRIEF NOTES ON PLANT DISEASES

SCLEROTINIA STEM BLIGHT ON STOCKS IN CALIFORNIA: Stem blight of stocks (Matthiola incana) due to Sclerotinia sclerotiorum was observed at Mission San Jose on February 14 where it was causing considerable loss in a commercial field planting grown for cut flowers. Sclerotia are formed in the pith cavity as well as externally. (M. W. Gardner, University of California. Feb. 16).

WHEAT RUSTS IN KANSAS: Leaf rust [Puccinia rubigo-vera tritici] and stem rust [P. graminis] that appeared in generous amounts on volunteer wheat in October were exceedingly hard to find after December 1, owing to low temperatures and dry conditions. (C. O. Johnston. Jan. 24. Cereal Courier 31: 7. Feb. 10, 1939).

CEREAL DISEASES IN CALIFORNIA: Subnormal rainfall for the greater part of California this crop year seems certain now. Precipitation from July 1 to date totals only 3.94 inches, whereas the normal for this period

is 10 inches. The indicated deficiency at Davis is quite characteristic of most sections of the State. We normally expect only 7 inches of rain during the remainder of the crop year.

Frosts have been very common this winter, although the minimum temperature has never fallen below 25°. There has been an abundance of sunshine. Nearly all cereal crops have been sown. Growth is not characteristic since plants are much shorter and darker green than normal. Tillering and root formation are satisfactory. Stem rust seems to be overwintering successfully on summer-germinated wheat and barley. Other diseases are less prevalent than usual at this season. (C. A. Suneson, Jan. 31. Cereal Courier 31: 9. Feb. 10, 1939).

NEW DISEASES OR NEW DISTRIBUTION REPORTED IN THE LITERATURE

DISCOVERY OF CITRUS CANKER IN NEW ZEALAND: W. D. Reid has reported the discovery of the presence of citrus canker caused by Bacterium citri in New Zealand. In a survey of the citrus districts made after the initial determination of the organism in lemon leaves from Kerikeri, Bay of Islands, in May 1937, the disease was found to be widespread in the North Island and was located in all other citrus-growing areas with the exception of those in Hawke's Bay and the South Island. In all cases infection was traced to citrus nursery stock introduced from one nursery. The manner in which it was introduced into New Zealand could not be determined; it had probably been present for not more than three seasons. The disease was confined to a few trees in isolated orchards, except in a small compact portion in Kerikeri, where in twelve affected orchards approximately three thousand trees were treated. Injury was relatively slight and the disease was found only on current season's growth, except for a few trees on which it occurred on older wood and which were promptly eradicated. The behavior of the disease under New Zealand conditions indicates that it will not assume the importance there that it has in other countries, and therefore that drastic eradication measures as carried out in the United States and in South Africa will not be necessary. The control program instituted includes severe pruning or "bare-poling" of all trees in orchards in which the disease has appeared, involving the removal of all tissues showing green coloration so that only the main limbs remain, together with the application of various sanitation practices, including spraying with lime-sulfur. The author stated that recent inspections had shown this treatment to be successful and that with its strict application there seemed to be no reason why the disease should not be eliminated from New Zealand. (Reid, W. D. Citrus-canker in New Zealand. New Zealand Jour. Sci. & Techn. 20: 55A-62A. June 1938).

A BACTERIAL DISEASE OF STOCKS IN NEW SOUTH WALES: A bacterial disease of stocks (Matthiola incana var. annua) not hitherto reported to

occur in New South Wales was observed in several localities and was responsible for heavy losses in some plantings. The disease is caused by an organism closely related to Bacterium campestre (Pammel) EFS. and is probably the same as the one recorded by Kendrick (Phytopath. 28: 12. 1938) from California. (Wilson, R. D. A bacterial disease of stocks. Jour. Austral. Inst. Agr. Sci. 4: 212-215. Dec. 1938).

PHYTOPHTHORA DISEASE OF HOPS IN NEW ZEALAND: Phytophthora cactorum has been shown to be the cause of a disease of hops known as black root-rot in the Nelson Province of New Zealand, according to R. M. Brien. This was believed to be the first record of the fungus on hops. (Brien, R. M. Black root-rot of hops. New Zealand Jour. Sci. & Techn. 20: 62A-64A. June 1938).